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Part B:
PCB Human Health Risk Assessment

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PART B: PCB HUMAN HEALTH RISK ASSESSMENT

1.0 INTRODUCTION

1.1 Overview of USEPA's Risk Assessment Approach

The companion to this paper, Part A, discussed the properties of PCB mixtures and how they behave once released into the environment. As discussed, the PCBs historically released at Navy installations have become highly weathered PCB mixtures. The unique properties of these complex weathered PCBs make human health assessments (HHRAs) more challenging than those conducted for other conventional non-PCB sites. However, all HHRAs comprise the following four steps:

- ***Step 1:*** *Data Assessment;*
- ***Step 2:*** *Exposure Assessment;*
- ***Step 3:*** *Toxicity Assessment; and*
- ***Step 4:*** *Risk Characterization.*

At most Navy installations, the two most difficult steps of the PCB HHRA will be the first and third steps, which involve sampling and analysis, and the assessment of toxicity of the weathered PCB mixture, which will be unique at each site. That is, each PCB will differ in: (1) The compositional diversity of different Aroclor originally released at the site, and (2) Weathering and differential partitioning of PCB mixtures released into the environment over time. For these reasons, U.S. EPA PCB human health risk assessment guidance (USEPA 1996) requires a tiered approach that explicitly focuses on the current site-specific composition of the weathered PCB mixture, rather than the original commercial Aroclor. In this approach, health risks are first estimated based on total PCB concentration (rather than type of Aroclor mixture) detected in each *environmental medium*. To these risks are added the carcinogenic risks associated with dioxin-like PCB congeners. The final cancer risk estimate is the sum of the two classes of PCB congeners—namely, dioxin-like and non-dioxin like PCBs—as well as risks from impurities in the original Aroclor mixtures, which will primarily be dioxin-like furans.

As discussed in Part A, PCB congeners released into the environment will partition into different environmental media (water, soil, air, animals, etc.) based on the chemical properties of each congener. This results in PCB congeners with different toxicities partitioning into different environmental media. Consequently, USEPA guidance specifically requires the use of 3 different toxicity values for three general groups of PCB congeners.

The following sections discuss the type of chemical data that is necessary to conduct a human health risk assessment and how that data is used. The final sections provide a hypothetical case study.

1.2 PCB Data Required for a HHR

PCB toxicity is a function of which congeners are present, not on the original composition when produced as an Aroclor. Since PCB undergoes weathering in the environment, Aroclor data is of limited use in a HHRA. PCB data necessary to conduct a HHRA must accurately represent exposure conditions at the site. At most sites where

Aroclor mixtures have been released, PCBs will be present, but typically in a composition markedly different from the original commercial mixtures. While it may be cost-effective to collect samples and conduct Aroclor analyses to screen sites, Aroclor data by itself will usually prove insufficient to precisely estimate human health risks. The composition of PCB mixtures changes over time through partitioning, chemical transformation, and preferential bioaccumulation; thus, Aroclor data can lead to either overestimating or underestimating PCB concentrations and concomitant risks. For this reason, Aroclor data should be used very carefully, and usually only for initial site screening. Additionally, Aroclor analysis will entail careful interpretation of laboratory chromatograms to ensure reported nondetect data truly represent a total absence of PCBs in a sample, rather than the subjective conclusion by the chemist that a "particular" Aroclor is not present. Conversely, the presence of two or more Aroclors can lead to overestimation of the total PCB concentration by double-counting groups of PCB congeners that are common to more than one Aroclor. Since the purpose of an HHRA is to estimate risks associated with exposure to PCBs as opposed to Aroclors (which may no longer be distinguishable), it is usually necessary to augment Aroclor analysis with both PCB homologue and PCB congener data. This is stressed by USEPA (USEPA 1996):

> *"Although PCB exposures are often characterized in terms of Aroclors, this can be both imprecise and inappropriate. Total PCBs or congener or isomer analyses are recommended."*

USEPA's concern about Aroclor data is consistent with the recommendations of the National Academy of Sciences, NRC for conducting PCB analyses. In their discussion of methods of analysis of PCBs, the NRC (2001) states:

> *"Unfortunately, the environmental weathering of Aroclors modulates mixture toxicity (Quensen et al. 1998). As such, carcinogenic risk-assessment guidelines recommend the calculation of congener-specific or total PCB data when available (EPA 1994c). Congener-specific analyses utilize the direct quantification of each unique PCB congener. The result is a precise description of PCB profiles, which can highlight physiological, spatial, and temporal changes that might not be apparent in Aroclor values."*

Some HHRAs have attempted to "improve" Aroclor analysis mathematically. In discussing some of the attempts to statistically "adjust" Aroclor data, the NRC (2001) states that even statistical manipulation cannot make up for the shortcomings in Aroclor data:

> *"Despite that, the Aroclor method does not adequately represent the concentrations found in weathered environmental samples. The discrepancies in the congener composition between the commercial mixture and real-world environmental exposures imply that the predictive value of studies based on commercial mixtures might be limited with respect to estimating risks from environmental exposure."*

In summary, individual PCB congener data provides the most flexibility for supporting environmental management decisions at PCB sites where Aroclors have been released. This also holds true for sites where PCBs have been generated *de novo* through combustion. At those sites, Aroclor analysis should not even be used for screening purposes, since only PCB homologue or congener data will provide the necessary chemical specificity to screen and characterize the site.

2.0 PCB TOXICITY

The toxicity of PCBs is supported by numerous peer-reviewed toxicity studies. Toxicity studies include *in vivo, in vitro,* and epidemiological studies for PCB mixtures (primarily involving commercial Aroclors), as well as for individual PCB congeners. The following section presents an overview of the important aspects of PCB toxicity and toxicity values that are used to estimate human health risks.

2.1 Non-Cancer Effects of PCB Mixtures

The noncancer health effects of PCBs are well known. PCBs are readily absorbed into the body from the gastrointestinal tract, skin, and lungs. PCBs initially concentrate in the liver, blood, and muscle, but are soon sequestered into fat tissue, where they have a long half-life, typically on the order of decades. PCBs are metabolized to biphenyls, biphenyldiols, and dihydrodihydroxybiphenyls, which are ultimately excreted in urine and feces. Animal studies reveal a considerable variation in equipotent doses between species of both animals and PCBs. In comparable studies, however, the more-chlorinated mixtures are more toxic than are the less-chlorinated ones. This trend predominantly holds between LD50 and carcinogenicity studies. In humans, the primary acute toxic effect of PCBs is chloracne—a unique and severe form of skin eruptations.

Chronic ingestion of PCBs causes "Yusho Disease," named after the town of Yusho, Japan, where an epidemic occurred when residents ate PCB-contaminated food for several months. Much of what we know about the toxic effects of PCBs in humans was reported in the Yusho studies.

Chloracne develops after a latent period, along with hyperpigmentation of skin areas, visual disturbances, gastrointestinal distress, jaundice, and lethargy. Infants born to exposed mothers have low birth weight and pigment blotches. Some of these effects, however, have been attributed to the chemically related polychlorinated dibenzofurans (CDFs), which are contaminant byproducts found in most complex mixtures of PCBs. Industrial exposure, which is generally limited to dermal contact, produces chloracne and, in severe cases, hepatotoxicity. PCBs produce reproductive toxicity based on results of the few animal studies; the Yusho incident; and, more recently, a similar incident in Taiwan. The systemic (noncancer) effects are represented by the reference dose (RfD). The RfD for Aroclor 1012 is 7E-5 mg/kg-day, based on the toxic effect of reduced birth weights. For Aroclor 1254, the RfD is slightly lower at 2E-5 mg/kg-day and is based on the toxic effects of ocular exudate (eye secretions), inflamed and prominent Meibomian glands, distorted growth of finger and toe nails, and decreased antibody (IgG and IgM) response to sheep erythrocytes.

2.2 Cancer Potential of PCB Mixtures

PCBs are class B2, or probable human carcinogens, based on the induction of liver tumors in experimental animals (EPA 1995). Unlike conventional risk assessments, where specific toxicity values are developed for individual chemicals, Aroclors are complex mixtures of PCB congeners that differentially partition into water, sediment, and fish. As a general rule, more highly chlorinated PCB congeners concentrate into media with high organic content, such as soil sediments and biological systems, while congeners with low chlorine content tend to be more volatile and also more soluble in water. As a general rule, more-chlorinated PCB congeners are more toxic than less-chlorinated PCBs.

2.2.1 USEPA's Tiered Toxicity Paradigm Used for Cancer Effects

USEPA PCB risk assessment methodology for estimating carcinogenic risks is based on differential partitioning of PCB congeners into different environmental media. It is based on the well-established partitioning processes that govern the fate and transport of PCBs, leading to an increase or decrease in toxicity in an individual environmental medium. That is, the toxicity of an environmental mixture is dependent on the type and concentration of PCB congeners that partition in that particular medium. The toxicity of environmental media is, therefore, only partly determined by the original Aroclor commercial mixture

Table B-1 presents USEPA's (1996) tiered toxicity paradigm based on partitioning of PCB congeners in *environmental media*. It is *not* based on the detection of different Aroclors in environmental media. The highest observed potency from these ranges is appropriate for food chain exposure, sediment or soil ingestion, and dust or aerosol inhalation. These are the pathways in which differential partitioning processes tend to increase risk associated with more-chlorinated toxic PCBs. Lower potencies are appropriate for ingestion of water-soluble congeners or inhalation of evaporated congeners, which are pathways where environmental processes tend to decrease risk. To the extent that drinking water or ambient air contains contaminated sediment or dust, the higher potency values would be appropriate, as congeners adsorbed to sediment or dusts tend to be of high chlorine content and persistence, especially for sediment or dust with high organic content.

Table B-1. PCB Toxicity Values For Environmental Media Based On Exposure Routes

HIGH RISK AND PERSISTENCE				
ED10	**LED10**	**Central Slope Factor**	**Upper-Bound Slope Factor**	**Exposure Pathways**
0.086	0.067	1	2	Food chain exposure Sediment or soil ingestion Dust or aerosol inhalation Dermal exposure, if an absorption factor has been applied to reduce the external dose Presence of dioxin-like, tumor-promoting, or persistent congeners in other media Early-life exposure (all pathways and mixtures)
LOW RISK AND PERSISTENCE				
ED10	**LED10**	**Central Slope Factor**	**Upper-Bound Slope Factor**	**Exposure Pathways**
0.38	0.27	0.3	0.4	Ingestion of water-soluble congeners Inhalation of evaporated congeners Dermal exposure, if no absorption factor has been applied to reduce the external dose
LOWEST RISK AND PERSISTENCE				
ED10	**LED10**	**Central Slope Factor**	**Upper-Bound Slope Factor**	**Exposure Pathways**
2.4	1.4	0.04	0.07	Congener or isomer analyses verify that congeners with more than 4 chlorines constitute less than 0.5% of total PCBs

Notes: ED10=Estimated dose associated with 10% increased incidence, in mg/kg-d;
LED10=95% lower bound on ED10, in mg/kg-d;
Central Slope=per mg/kg-d, computed as 0.10/ED10 and rounded to one significant digit;
Upper-Bound Slope=per mg/kg-d, computed as 0.10/LED10 and rounded to one

One step that should not be ignored in HHRA at PCB sites (which is overlooked at non-PCB sites) is the use of central-estimate slope factors, presented in Table B-1, to estimate the central tendency exposure (CTE) risk. These are derived by linear extrapolation from points representing the effective dose in 10 percent of animals (ED10s), which are mathematically derived with three reference points. Central-estimate slope factors are used to estimate a typical individual's risk, while upper-bound slope factors assure that this risk is not likely to be underestimated if the underlying model is correct. Both slope factors should be used in the HHRA to estimate CTE and reasonable maximum exposure (RME) risks.

2.2.2 Dioxin-Like PCB Congeners

Certain PCB congeners have been identified as having dioxin-like toxicity. This designation was made based on similarities in structure, biochemical activity, and the ability of these PCB congeners and of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) to bioaccumulate.

In evaluating cancer risk for dioxin-like PCB congeners, the total 2,3,7,8-TCDD equivalent concentration is calculated for each sample based on the analytical results of the PCB congeners. This calculation is performed by multiplying the concentration of each PCB congener by its corresponding 2,3,7,8-TCDD TEF and then summing the results. This summed result is then used in exposure calculations as the concentration term.

The slope factor for 2,3,7,8-TCDD is then applied to the average daily dose calculations to evaluate cancer risk for dioxin-like PCB congeners. The cancer slope factor for 2,3,7,8-TCDD is 150,000 mg/kg-day (EPA, 1997).

While this cancer risk calculation provides a point of comparison to the cancer risks calculated using slope factors derived for PCB mixtures, it is uncertain for the following reasons:

- PCBs are typically found as mixtures in environmental media, not in the pure congener form, and the toxicity of mixtures is typically different than that of a pure compound. Limiting the potency of environmental mixtures of PCBs to the range observed for commercial mixtures reflects a decision to base potency estimates on experimental results, however uncertain, rather than apply safety factors to compensate for lack of information (EPA, 1996a).
- Only a few congeners have undergone toxicity testing and none in long-term carcinogenesis studies (EPA, 1996a).
- EPA considered all cancer studies (which used commercial Aroclor mixtures only) and developed a range of dose-response slopes (EPA, 1996a). The highest PCB slope factor derived by EPA for mixtures is close to 5 orders of magnitude lower than the slope factor for 2,3,7,8-TCDD (used for dioxin-like PCBs).
- New PCB Slope factor has actually decreased from 7.7 per mg/kg-day to a maximum of 2 per mg/kg-day. This incorporates both human and animal data.
- Despite a new Dioxin Reassessment the USEPA is not consistently requiring that the regulated community use the "new Dioxin Slope Factor" which is at

least 1 order of magnitude higher that the 150,000 mg/kg-day-1 that is currently being used.

- The TEF approach for Dioxin-Like PCB congeners is based on structural-activity/similarity with 2,3,7,8-TCDD Congeners. The science is weak and is contrary to the PCB Reassessment which recommended a decrease in the Slope factor for PCB mixtures. It is important to note that these Dioxin-like PCBs congeners are included in " Total PCBs" so there is a contradiction as to how the Total PCB slope factor can decrease when at the same time USEPA and others are stating that the PCB risks are being underestimated because we are not evaluating Dioxin-like PCB congeners.

In addition, as of the time of this writing (November 2005) the USEPA Integrated Risk Information System (IRIS) database states, "*when congener concentrations are available, the slope factor approach* [the tiered approach shown in Table B-1] *can be supplemented by analysis of dioxin TEQs to evaluate dioxin-like toxicity. Risks from dioxin-like congeners (evaluated using dioxin TEQs) would be added to risks from the rest of the mixture(evaluated using slope factors applied to total PCBs reduced by the amount of dioxin-like congeners).*" This statement implies that there is no requirement to evaluate DL-PCBs. If congener concentrations are available, the recommended approach (the slope factor approach) can be supplemented by analysis of dioxin like toxicity. Therefore, at this point in time, human health risks associated with dioxin-like PCB congeners do not have to be added to the risks calculated for total PCBs at the site. Instead due to the uncertainties mentioned above, the human health risk associated with dioxin-like PCBs, if calculated, should only be included as an uncertainty in the uncertainty section of the human health risk assessment report, until these uncertainties are resolved.

Although only a small group of 13 PCB congeners produce dioxin-like effects, they should only be evaluated when present in any environmental medium, if the analytical method used has the ability to resolve or separate many PCB congeners. When potential carcinogenicity is of concern to consumers of higher trophic level organisms, a highly sensitive and specific congener method is required. For these types of assessments, the most reliable analytical method is 1668 in a qualified laboratory with experienced staff.

Table A-3 (in Part A) presents a cost-effective sampling and analysis approach for determining the likelihood of dioxin-like PCBs being present at the site. Due to the high cost of PCB congener analysis for this group (using *EPA Method 1668a*), preliminary analysis using Aroclor and/or PCB homologue analysis is warranted.

It must be noted that no single specific analytical approach will answer all questions or address all concerns. Each program must identify its data quality objectives, typical end users and available resources before deciding on an analytical approach for assessment of environmental impact from PCBs contamination. Depending on the objectives, it is not usually necessary to conduct extensive PCB congener analysis. It may only be necessary to conduct PCB congener analyses on enough samples to establish a mathematical relationship with the total PCB concentration, assuming one exists. When there is a strong correlation between the concentration of dioxin-like congeners and total PCBs, PCB congener analysis can be stopped, and the concentration of dioxin-

like congener concentration in each sample can be roughly estimated based on the total concentration of PCBs using Aroclor or PCB homologue analysis.

As noted in Part A, all commercial Aroclor mixtures contain varying amounts of PCB dioxin-like congeners. The concentration of each of the 13 dioxin-like PCB congeners has been determined for commercial Aroclor mixtures by Schwartz et al. (1993) and is present in Table B-2. Although these concentrations can be used as a rough estimate of the individual PCB congener concentrations in the original commercial mixture, they may not represent site-specific conditions due to weathering. That is, weathering may result in a relative "enrichment" of dioxin-like congeners based on Aroclor data because non-dioxin PCB congeners may preferentially migrate to other media or be degraded. The relative concentration of dioxin-like congeners in soil and sediment would then represent a higher percentage of the weathered Aroclor mixture.

Table B-2. Dioxin-Like PCB Congener Concentrations In Aroclor Mixtures

PCB CONGENER NUMBER	CONCENTRATION OF DIOXIN-LIKE PCB CONGENERS IN AROCLOR MIXTURES			
	Aroclor 1242	**Aroclor 1248**	**Aroclor 1254**	**Aroclor 1260**
PCB 61	159	305	<4	<55
PCB 77	1700	2990	200	<61
PCB 105	2670	13600	32100	245
PCB 114	328	1829	2460	28
PCB 116	3620	19900	75800	4470
PCB 123	63	260	560	<20
PCB 126	16	38	88	<52
PCB 128	274	1740	23900	17400
PCB 138	1090	6670	116000	152000
PCB 158	36	387	7610	2940
PCB 157	19	101	3410	NA
PCB166	4	20	211	<16
PCB 167	20	185	4390	1900
PCB 169	<12	>2	<3	<42
PCB 170	39	702	7910	35000
PCB 189	<7	11	268	885

Notes:
Reference: Schwartz et al. 1993
< : Denotes less than detection limit
Congeners in parts per million (µg/g Aroclor)

Dioxin-like PCB toxic effects are identical to those produced by 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as stated in USEPA (1996) guidance and must be included in the HHRA:

> *"When assessing PCB mixtures, it is important to recognize that both dioxin-like and nondioxin-like modes of action contribute to overall PCB toxicity (Safe, 1994; McFarland and Clarke, 1989; Birnbaum and DeVito, in press[1995]). Because relatively few PCB congeners are dioxin-like, dioxin equivalence explains only part of a PCB mixture's toxicity."*

The importance of dioxin-like congeners is also emphasized by the National Academy of Sciences (NRC 2001):

> *"The non- and mono-ortho-substituted PCBs are of particular concern, because these congeners can assume a planar or nearly planar conformation similar to that of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Safe, 1990; Giesy et al., 1994a; Metcalfe and Haffner, 1995) and have toxic effects similar to TCDD."*

It should be noted that CDFs are also likely to be present at sites where Aroclors have been released because they are typical contaminants of Aroclor mixtures. The will likely also be present along with dioxins at PCB sites where PCBs are thought to be generated *de novo*.

2.2.3 Toxicity Equivalent Factors for Dioxin-like PCBs, CDFs, and CDDs

Evaluating dioxin-like PCB congener data in an HHRA involves assigning congener-specific toxicity equivalency factors (TEFs) to each of the 13 PCB congeners. The TEF values developed by Ahlborg et al. (1994) and USEPA (1998) are based on the toxicity of the archetypical reference standard, TCDD, which is assigned a TEF of 1.0. PCB congeners have TEF values ranging from 0.1 to 0.00001, which are presented in Table B-3. Calculating the toxic equivalency (TEQ) of a mixture of PCB congeners (which is used directly in the HHRA) simply involves multiplying the concentration of individual congeners by their respective TEFs. The sum of the TEQ concentrations for the individual congeners is the TEQ concentration for the mixture, which is multiplied by the slope factor for TCDD in the risk assessment to calculate PCB dioxin-like carcinogenic risks.

Table B-3. USEPA TEF Values For Dioxin-Like PCBs

PCB Dioxin-Like Congener	PCB Congener Number	Toxicity Equivalent Factors (TEFs)
3,3',4,4'-Tetrachlorobiphenyl	PCB 77	0.0005
2,3,3',4,4'-Pentachlorobiphenyl	PCB 105	0.0001
2,3,4,4',5-Pentachlorobiphenyl	PCB 114	0.0005
2,3',4,4',5-Pentachlorobiphenyl	PCB 118	0.0001
2',3,4,4',5-Pentachlorobiphenyl	PCB 123	0.0001
3,3',4,4',5-Pentachlorobiphenyl	PCB 126	0.1
2,3,3',4,4',5-Hexachlorobiphenyl	PCB 156	0.0005
2,3,3',4,4',5'-Hexachlorobiphenyl	PCB 157	0.0005
2,3',4,4',5,5'-Hexachlorobiphenyl	PCB 167	0.00001
3,3',4,4',5,5'-Hexachlorobiphenyl	PCB 169	0.01
2,2',3,3',4,4',5-Heptachlorobiphenyl	PCB 170	0.0001
2,2',3,4,4',5,5'-Heptachlorobiphenyl	PCB 180	0.00001
2,3,3',4,4',5,5'-Heptachlorobiphenyl	PCB 189	0.0001

Although the *concentrations* of dioxin-like congeners at PCB sites may be significantly lower than other non-dioxin-like PCBs, they may pose greater health risks because they are potent carcinogens and have a much higher toxicity value. Carcinogenic slope factors for some dioxin-like PCB congeners are orders of magnitude higher than <u>non</u>-dioxin-like PCB congeners.

It is noteworthy that all dioxin-like PCB congeners have 4, 5, 6, or 7 chlorines. Consequently, if PCB homologue analysis indicates those groups are not present in any samples at the site, it is not necessary to pursue PCB congener analysis (which is considerably more expensive than Aroclor or homologue analysis) for the presence of dioxin-like PCBs, as indicated in Part A, Table A-3. When homologue analysis indicates they are present, it may be necessary to analyze for all PCB dioxin-like congeners, as well as for CDFs. Where *de novo* generation of PCBs is suspected, it may be necessary to also analyze for dioxins.

There are 75 possible chlorinated dibenzo-p-dioxins (CDD) and 135 CDF congeners. However, only 7 of the 75 CDD congeners have been to shown to produce "dioxin-like" toxicity. These are the dioxins that have chlorine substitutions in (at minimum) the 2,3,7,and 8 positions. Likewise, only 10 of the 135 possible CDF congeners produce dioxin-like toxicity and, again, are those that are chlorinated in 2,3,7, and 8 positions. Table B-4 presents TEF values that have been developed by USEPA for dioxin-like CDDs and CDFs.

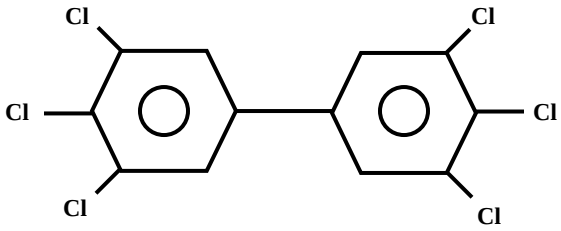
Table B-4. USEPA TEF Values For Dioxins And Furans

DIOXIN-LIKE CONGENERS	TOXICITY EQUIVALENT FACTORS (TEFS)
Dioxins	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	0.5
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
1,2,3,4,6,7,8,9-OCDD	0.001
Furans	
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
1,2,3,4,6,7,8,9-OCDF	0.001

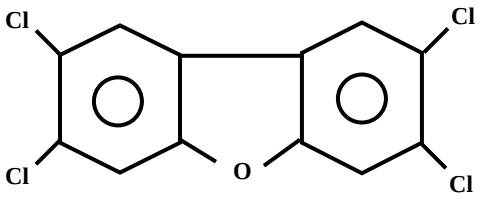
Note: TEFs are based on the relative toxicity of 2,3,7,8-TCDD

As discussed in Part A, the reason all dioxin-like compounds share the same toxic properties is because they are coplanar and produce their toxic effect by binding to the Ah-receptor in target cells in the body. Figure B-1 illustrates this molecular structural similarity shared by all dioxin-like compounds. As shown, PCB 169 is a non-ortho coplanar molecule. In other words, the two biphenyl rings resemble a pair of eyeglasses—where they assume a flat or planar shape, similar to the rigidly planar shape of 2,3,7,8-tetrachlorodibenzofuran and 2,3,7,8-tetrachlorodibenzo-p-dioxin. To produce the specific toxicity of dioxin-like compounds, they must assume this planar shape to bind effectively to the Ah-receptor, which triggers the toxic reaction.

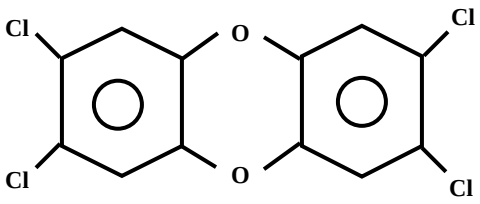
Figure B-1. structural similarity of dioxin-like compounds: pcbs, cdds, and cdfs



3,3',4,4',5,5'-Hexachlorobiphenyl – PCB 169



2,3,7,8 Tetrachlorodibenzofuran



2,3,7,8 Tetrachlorodibenzo-p-dioxin (TCDD)

At sites where the presence of CDFs and CDDs are suspected, *EPA Method 1613* should be used to quantify the concentrations of the 17 dioxin-like congeners. Human health risks for these 17 congeners are calculated in exactly the same manner as for the 13 PCB dioxin-like congeners and, finally, these are summed to estimate total carcinogenic risks for the site.

2.2.4 Overview of Existing USEPA Dioxin Database

USEPA recently completed a comprehensive reassessment of dioxin-related human health effects entitled *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*. It is expected that this report will be released for public comment in December 2002. This document will be updated to include new toxicity information on TCDD because it will have a major impact on how the PCB HHRAs are conducted and the risks estimated for Navy installations. For example, the carcinogenic slope factor for TCDD is expected to increase, which will have a direct effect on risks estimated for dioxin-like PCBs since PCB TEF values are directly based on TCDD.

3.0 SAMPLING AND ANALYSIS FOR HHRAS

Sampling and analysis to estimate human health risks at PCB sites should follow the general guidelines developed for all HHRAs. The first step is to develop a conceptual site model that defines the area over which exposure will occur and the type of exposure routes anticipated for the same individual.

The ultimate goal of sampling and analysis for PCB HHRAs is two-fold. The first objective is to determine the *total* PCB concentration in a particular sample and the second is to quantify the concentration of dioxin-like PCB congeners (if they are present). Knowledge of how PCB congeners differentially partition among all the environmental media should guide *where* samples are collected, the *number* of samples needed to accurately estimate the exposure point concentration, as well as the *type* of PCB analysis performed on the sample. For example, due to the hydrophobic nature of PCBs, they are not typically detected in water samples—either surface water or groundwater. Therefore, except for a few screening level analyses that can be conducted with simple Aroclor or PCB homologue analysis, few water samples are needed for a HHRA. The exception to this rule is when the water is turbid with organic-containing suspended particles. When PCBs are detected in water, it usually indicates a recent release or high levels of suspended sediments to which PCBs are adsorbed. In most sites, however, it is not necessary to focus significant sampling efforts on water samples. Certainly, expensive PCB congener analysis should be avoided.

In contrast, environmental media with high organic content should be a major focus of sampling as it provides a reservoir for PCBs, particularly for highly chlorinated PCBs, which include the most toxic PCB congeners. Additionally, bioaccumulated PCBs appear to be more toxic than commercial PCBs and are more persistent in the body. For this reason, selecting the correct *type* of PCB analysis is important. Ingestion of fish or other marine species (when present at the site) is central to most HHRAs. For this reason, samples collected from biological tissues should always be analyzed for PCB congeners because biological systems will bioaccumulate dioxin-like PCB congeners. The congener-specific selective bioaccumulation into fish will most often result in the congener profile not resembling the original Aroclor that was released, which can lead to false negative conclusions regarding the presence of PCBs in fish.

Finally, as noted in previous sections, sampling and analysis at PCB sites should always quantify levels of chlorinated dibenzofurans. As previously noted, CDFs are contaminants detected in relatively high concentrations in commercial Aroclor mixtures. Consequently, CDFs would be released together with PCBs and should be quantified in

all pertinent environmental media containing high organic content and in all biological tissues. Additionally, at sites where PCBs have been formed *de novo* through combustion, CDDs should be analyzed.

3.1 Non-Detect Data

As mentioned previously, one flaw in using Aroclor data in HHRAs is the potential for risks to be overestimated if Aroclor concentrations are simply summed. When two or more Aroclors are identified, "double counting" congeners common to different Aroclors may occur.

In addition to this potential problem, one important modification in treating nondetect Aroclor data should be made in PCB HHRAs: a proxy value of one-half the detection limit *should not* be substituted for the nondetect datum. For chemicals other than PCBs, it is conventional to assign a value of one-half the detection limit for nondetect values under the presumption that the chemical could be present in the sample at a concentration just below the detection limit. Using one-half the detection limit has been justified on the theory that the chemical, in theory, could be present in a sample at a concentration ranging from zero (completely absent) to just below the detection limit. One-half the detection limit is thought to be a mathematical "compromise." However, when Aroclor data is used to estimate the total PCB concentration at the site and all potential Aroclors are analyzed (e.g. 1016, 1242, 1248, etc.), the assumption that all nondetect Aroclors are truly present at one-half the detection limit is flawed for two reasons. The first is that identification of different Aroclors in a particular sample is not (usually) limited by detection limits, but rather by the presence or absence of a particular Aroclor fingerprint identified in the chromatogram (based on 5 or 6 points of identification). The absence of a fingerprint, while subjective, is a good indication that a particular Aroclor is simply not present. Assuming it is present just below the detection limit and using one-half the detection limit as a proxy value cannot be justified.

The second reason that assigning one-half the detection limit to all nondetect Aroclors in a particular sample is incorrect is because it will result in unintended "double counting" of those congeners that are common to different Aroclors.

These problems further illustrate the confounding factors involved when Aroclor data is used to quantify PCB releases at Navy installations. Aroclor analyses should be limited to screening sites for the presence or absence of PCBs. It is unreliable for determining the nature and extent of contamination or for quantifying human health risks. For the above reasons, PCB homologue analyses are superior to Aroclor analyses and should be considered a cost-effective alternative.

3.2 Analyzing for Background Conditions

Background conditions should always be defined for sites. Navy releases must be distinguished from ubiquitous anthropogenic releases or unrelated point sources. However, a robust and detailed background analysis must be conducted with PCB congener data. Although the complexity of Aroclor mixtures makes PCB investigations more difficult in general, it actually provides an advantage in background analyses. That is because each PCB released into the environment will have a unique fingerprint based on the relative ratios of each individual PCB congener. Each unique PCB fingerprint

results from the degree of weathering the Aroclor has undergone since the original PCB mixture (Aroclor) mixture was released.

When PCBs are detected at Navy installations, the project team will be faced with making a determination of whether the PCBs are the result of a Navy release or simply represent anthropogenic background conditions. Alternatively, a release by the Navy may have added to the background levels. To make these determinations, the project team should conduct statistical analysis based on the relative ratios between paired PCB congeners using linear regression or conduct principal component analysis. While it is outside the scope of this document to provide details for conducting these statistical analyses, they should always be performed at PCB sites. Furthermore, due to the aforementioned problems with Aroclor analysis, Aroclor data should never be used to quantify background conditions or conduct a background analysis.

4.0 CASE STUDY EXAMPLE

Conventional methods, similar to those used at all non-PCB sites, are used to calculate noncancer health hazards. The RfDs, which are presented in Section 1.2.1, are simply divided by the estimated average daily dose. However, the methodology for estimating carcinogenic risks associated with total PCBs and dioxin-like PCBs (and CDFs) is markedly different from conventional approaches in which chemical-specific toxicity values are directly multiplied by the lifetime average daily dose. For PCB mixtures, toxicity values used to estimate risks are based on exposure routes. The following sections present a hypothetical PCB site at a Navy installation to illustrate the steps of a PCB risk assessment, highlighting major differences from non-PCB HHRAs.

4.1 Site Background

A hypothetical Navy installation located next to a river was investigated as a possible PCB site where Aroclors were suspected of being released during maintenance of electrical transformers. The project team followed the sampling and analysis steps outlined in the flow chart in Part A,Table A-3 to implement a cost-effective sampling and analysis regime.

In the initial screening phase, Aroclor analysis indicated PCBs were indeed present. The project team decided remedial decisions should be based on human health risks (rather than regulatory standards). Consequently, this decision triggered PCB homologue analysis, and the shift in sampling analysis was implemented in the next sampling round. The results of the homologue analysis indicated dioxin-like PCBs might be present, which then triggered subsequent PCB congener analysis in soils. CDFs analysis was also conducted on select media based on knowledge of environmental partitioning processes.

4.2 Data Assessment

Table B-5 presents a summary of total PCB concentrations determined for different environmental media. (Note: For the sake of brevity, only one sample is presented. For real Navy sites, all PCB data representing contaminated areas for all environmental media would be averaged to represent the exposure point concentration for each environmental medium.) Due to the problems inherent in Aroclor analyses, total PCB concentrations were determined with homologue analysis.

Table B-5. Example Site: Summary For Total PCBs

ENVIRONMENTAL MEDIUM –	TOTAL PCB CONCENTRATION
SOIL	5.0 mg/kg
VAPOR	0.01 µg/cu.m.
GROUNDWATER	5 µg/L
FISH	110 µg/kg

Note: Total PCB is based on homologue analysis.

Preliminary PCB homologue data indicated that dioxin-like PCB and CDF congeners might be present in soils. This finding triggered PCB congener analysis, which was conducted using *USEPA Method 1668.* CDFs were also analyzed using *USEPA Method 1613.* The results are presented in Table B-6.

Table B-6. Example Site Data:
Data Summary For Dioxin-Like PCBs and CDFs In Soil

DIOXIN-LIKE PCB AND FURAN CONGENERS IN SOIL	CONCENTRATION (µg/kg)	TEF	TEQ
PCB			
PCB 77	0.346	0.0005	0.000173
PCB 105	0.522	0.0001	0.000052
PCB 114	0.053	0.0005	0.000027
PCB 118	0.855	0.0001	0.000086
PCB 123	0	0.0001	0
PCB 126	0.004	0.1	0.0004
PCB 156	0.196	0.0005	0.000098
PCB 157	0.02	0.0005	0.00001
PCB 167	0.079	0.00001	0.000001
PCB 169	0.0002	0.01	0.000002
PCB 170	1.264	0.0001	0.000126
PCB 180	2.647	0.00001	0.000026
PCB 189	0.025	0.0001	0.000003
CDFs			
2,3,7,8-TCDF	0.0448	0.1000	0.0045
1,2,3,7,8-PeCDF	0.026	0.0500	0.0013
2,3,4,7,8-PeCDF	0.031	0.5000	0.0155
1,2,3,4,7,8-HxCDF	0.179	0.1000	0.0179
1,2,3,6,7,8-HxCDF	0.0592	0.1000	0.0059
1,2,3,7,8,9-HxCDF	0.0076	0.1000	0.0008
2,3,4,6,7,8-HxCDF	0.0667	0.1000	0.0067
1,2,3,4,6,7,8-HpCDF	0.352	0.0100	0.0035
1,2,3,4,7,8,9-HpCDF	0.0536	0.0100	0.0005
1,2,3,4,6,7,8,9-OCDF	0.3536	0.0010	0.0004
TOTAL DIOXIN-LIKE CONGENER TEQ			**0.058004**

Based on fate and transport processes involving differential partitioning of individual congeners, dioxin-like PCB congener analysis was also conducted for fish samples (samples were also analyzed for CDFs, which will likely be present in the same percentage as was detected in soil samples; however the CDF data was not available at the time this example was prepared). The dioxin-like PCB congener data for fish are presented in Table B-7.

Table B-7. Example Site:
Data Summary For Dioxin-Like PCBs In Fish

DIOXIN-LIKE PCB AND FURAN CONGENERS IN FISH	CONCENTRATION (µg/kg)	TEF	TEQ
PCB 77	2.1	0.0005	0.0011
PCB 105	14	0.0001	0.0014
PCB 114	1.8	0.0005	0.0009
PCB 118	54	0.0001	0.0054
PCB 123	1.4	0.0001	0.0001
PCB 126	0.14	0.1	0.014
PCB 156	5	0.0005	0.0025
PCB 157	1.1	0.0005	0.0006
PCB 167	7.7	0.00001	0.0001
PCB 169	0.0068	0.01	0.0001
PCB 170	0	0.0001	0
PCB 180	0	0.00001	0
PCB 189	0.28	0.0001	0
TOTAL			**0.0264**

4.3 Exposure Assessment

A conceptual site model was prepared to identify potential pathways for human exposure. Complete pathways include the following:

- Vapor inhalation;
- Soil Ingestion;
- Drinking water; and
- Fish Ingestion.

Potential receptors are adult receptors (that weigh average of 70 kg) that may be exposed to different environmental media. It is assumed that a person will consume an average of two 105-g portions of local fish each week. This person will spend most of his time in the area, breathing 20 cubic meters of air and drinking 2 liters of water, on average, each day. This person will also inadvertently ingest 100 milligrams of soil a day. The exposure duration is anticipated to last for 30 years, with a representative lifespan of 70 years. As shown in Table B-5, PCB homologue analysis indicated an

average concentration of 5.0 mg/kg in soil, 0.01 g/cubic meters in ambient air, 5 μg/liter in drinking water, and 110 μg/kg in the edible portion of local fish.

The lifetime average daily dose (LADD) is an estimate of the daily amount of ingested chemical. The LADD at PCB sites is calculated in a similar manner to the LADD calculated for non-PCB sites. That is, the LADD is calculated as the product of chemical concentration (C), intake rate (IR), and exposure duration (ED), divided by body weight (BW) and lifetime (LT). Table B-8 presents the exposure assumptions and the calculated LADD for each exposure pathway based on the exposure point concentration (EPC).

Table B-8. Example Site:
Calculating The Lifetime Average Daily Dose (LADD)

EXPOSURE ROUTE	EPC	I R	ED	BW	LT	LADD
TOTAL PCB						
SOIL INGESTION	1.1 mg/kg	100 mg/d	30 yr	70 kg	70 yr	5.2E-7 mg/kg-d
VAPOR INHALATION	0.01 μg/cu.m.	20 m /d 3	30 yr	70 kg	70 yr	1.2E-6 mg/kg-d
GROUNDWATER INGESTION	5 μg/L	2 L/d	30 yr	70 kg	70 yr	6.1E-5mg/kg-d
FISH INGESTION	110 μg/kg	30 g/d	30 yr	70 kg	70 yr	2.0-5 mg/kg-d
DIOXIN-LIKE CONGENERS						
SOIL INGESTION	5.8E-1	100 mg/d	30 yr	70 kg	70 yr	2.7E-7mg/kg-d
FISH INGESTION	2.6E-1	30 g/d	30 yr	70 kg	70 yr	4.8E-9 mg/kg-d

Notes:
LADD = Lifetime Average Daily Dose
LADD = C × IR × ED / (BW × LT)
Where:
EPC= Exposure Point Concentration
IR= Ingestion Rate
ED= Exposure Duration
BW= Body Weight
LT=Lifetime

4.4 Toxicity Assessment and Calculating Risk

Partitioning, transformation, and bioaccumulation govern how the original composition of the Aroclor weathers. Highly toxic congeners have an affinity for some environmental media and not for others. This is why different carcinogenic slope factors are used for *different environmental media*. Risks should never be estimated based on the type of Aroclors detected in each environmental media. The only exception to this rule is when Aroclors have recently been released (within a year) and weathering has not yet occurred. (When it can be shown that the Aroclor release is recent and significant

weathering has not occurred, it may be possible to rely solely on Aroclor analysis and associated toxicity values.) For past releases, it should always be assumed that Aroclors have undergone weathering and that PCB congeners are in dynamic equilibrium between different environmental media.

Vapor inhalation is associated with "low risk" because evaporating congeners tend to have low chlorine content and low toxicity. In addition, low chlorine-content PCB congeners are eliminated from the body relatively quickly. Thus, the low end of the carcinogenic range (upper-bound slope of 0.4 per mg/kg-d) is used for vapor inhalation. Similarly, ingesting drinking water would expose the receptor to the water-soluble fraction of the mixture of PCB congeners and, again, they would be "low-risk" congeners because they would have low chlorine content and be quickly eliminated from the body. However, if ambient air or drinking water contained significant amounts of contaminated resuspended dust or sediment, respectively, the high-end potency values should be used because these media would then contain adsorbed congeners with high chlorine content that are more toxic and persistent.

Human exposure through the food chain is associated with "high risk" because fish, in this example, selectively accumulate congeners with high chlorine content, which are more toxic and persist in the body for long periods of time. In this example, an upper-bound slope of 2 mg/kg-d is used for fish ingestion. Likewise PCBs in soil would have higher chlorine content and would require the same toxicity value. Table B-9 presents the calculated risk for each exposure pathway.

Table B-9. Example Site:
Calculating Carcinogenic Risks

EXPOSURE ROUTE	LADD	CARCINOGENIC SLOPE FACTOR	CARCINOGENIC RISK
TOTAL PCB			
Soil Ingestion	4.7E-7 mg/kg-d	2 mg/kg-d	1E-6
Vapor Inhalation	1.2E-6 mg/kg-d	0.4 mg/kg-d	4.8E-7
Groundwater Ingestion	6.1E-5 mg/kg-d	0.4 mg/kg-d	2.4E-5
Fish Ingestion	2.0E-5 mg/kg-d	2 mg/kg-d	4.0E-5
DIOXIN-LIKE CONGENERS			
Soil Ingestion	2.7E-8 mg/kg-d	150,000 mg/kg-d	4.2E-3
Fish Ingestion	4.8E-9 mg/kg-d	150,000 mg/kg-d	7.2E-4

Note: Risk = LADD × Carcinogenic Slope

The risks indicate that soil and fish ingestion pose the greatest carcinogenic risk. As indicated, while total PCBs pose risks that fall within the discretionary risk range (1E-6 to 1E-4) where remediation may not be warranted, the risks associated with dioxin-like

PCB and CDF congeners are approximately two orders of magnitude higher. This example HHRA highlights the importance of analyzing for dioxin-like compounds. Without it, the site might not be remediated and continue to pose unacceptable risk.

5.0 CONCLUSIONS AND RECOMMENDATIONS

The requirements for sampling and analysis for determining the nature and extent of contamination are significantly different from the type and amount of data necessary to conduct an HHRA. While Aroclor analysis *may* be sufficient to a screen site for contamination, with very few exceptions it will not provide the data necessary to fully characterize risks associated with complex weathered PCB mixtures. Indeed, Aroclor analysis may ultimately confound the investigation and subvert the goals of the project team. Although Aroclor analysis was routinely practiced in the past, more sophisticated and detailed PCB homologue and congener analysis should form the core of information and data used directly in the risk assessment. Many laboratories now routinely perform homologue and congener analysis at very low detection limits. As illustrated in the example case, Aroclor data should not be used in the risk assessment.

There are two categories of PCB data that must be available to risk assessors before they can accurately estimate risk at Navy PCB sites:

- Total PCB Concentrations in Each Environmental Media; and
- The Concentration of Each Dioxin-Like PCB, CDF, and CDD Congener in Each Environmental Media.

For this reason, risk assessors must be involved at every stage of the investigation to ensure the correct samples are being collected and analyzed. Although this is true for all sites, it is particularly important for PCB sites because Aroclor, homologue, and congener data cannot be pooled like data for individual chemicals at non-PCB sites.

Homologue or congener analysis should always be used to quantify total PCB concentrations. PCB and CDF congener analysis should always be performed when warranted, as suggested by homologue data. Aroclor data should never be used in HHRAs, as it is not scientifically defensible for the purpose of estimating risk. However, as discussed in Part A, Aroclor data can be used to screen sites and in developing a site conceptual model.

Background analysis should always be conducted at PCB sites and always be based on PCB congener data. This information will provide a unique fingerprint to determine whether a Navy release has indeed occurred, as well as the extent of the release.

Finally, the HHRA should be based on PCB homologue and congener data. Aroclor data should never be the sole basis of an HHRA. At many sites, dioxin-like PCBs and CDFs will pose risks greater than those posed by total PCBs.

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